decreases in central venous pressure and heart rate, listed in the same table, all of which are consistent with a decrease in cardiac output in the absence of reduced contractility.

With respect to Dr Krohn's comments regarding the limitations associated with the application of the widely used standard pulmonary vascular resistance (PVR) equation, we are in agreement that this method of PVR calculation is imperfect for the reasons he has enumerated. However, Dr Krohn seems to confuse PVR (affected by pulmonary vascular properties) with right ventricular afterload (affected additionally by left atrial pressure, LAP). Pulmonary vasodilation may reduce PVR while raising LAP, thus leaving PAP and right ventricular afterload relatively unchanged.[1] This ought not be viewed as misleading; rather it is simply a natural consequence of the right and left sides of the hearts being connected in series. Furthermore, inasmuch as previous clinical and experimental work have already well established the pulmonary vasodilatory effects of iNO, this was not the focus of our experiment. Rather, since clinically and experimentally observed increases in LAP may be due to a decrease in PVR and/or alterations in ventricular function, we sought to establish whether iNO affects left ventricular systolic or diastolic properties, as described above. We found that the increase in LAP was not due to depression of ventricular function, but was simply a consequence of pulmonary vasodilation.

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12/8/93302

Deep hypothermic circulatory arrest and retrograde cerebral perfusion

To the Editor:

I was disturbed when I read the article by Okita and associates concerning deep hypothermic circulatory arrest (DHCA) and retrograde cerebral perfusion (RCP) in patients undergoing operations on the aortic arch in the January 1998 issue of the Journal (1998;115:129-38). I have several major concerns about the paper. The most important concern relates to the fact that the authors are describing a relatively new technique of cerebral protection, namely RCP, and yet they have failed to include among their investigators or authors any neurologists or psychologists. There is no statement in the methods section of the paper regarding complete neurologic examination of their patients and whether this was performed before and after the operations. My own experience would suggest that a simple retrospective chart review relying on observations by surgical residents is inadequate to detect subtle focal neurologic deficits. For example, how many of these patients had careful assessment of their visual fields? How many patients had their postoperative fine motor skills compared with their preoperative fine motor skills? In fact, even a neurologic examination will not detect many of the cognitive deficits that can only be identified by careful psychometric testing. In the absence of such testing, it is unjustified for the authors to make the statement: "We empirically consider that a DHCA + RCP period of up to 80 minutes under a nasopharyngeal temperature of 18°C is safe." This statement is all the more questionable after viewing Fig 1, which demonstrates that patients who underwent a combined DHCA + RCP time of 70 to 80 minutes had an incidence of death or delirium that was greater that 50%. The authors state: "Postoperative transient delirium has been defined as a transient minor neurologic deficit such as disorientation and character change with no neurologic sequelae." This statement bears resemblance to the previously often heard statement within pediatric cardiac surgery that seizures in the early postoperative period in infants are of no long-term significance. Our prospective randomized trial of DHCA[1] has demonstrated that perioperative seizures do indeed have an association with subsequent impairment, as assessed by our developmental psychologists using comprehensive testing at age 1 year and 4 years with ongoing studies of this same cohort at age 8 years.

Until clinical studies have been undertaken with careful preoperative and postoperative neurologic examination by neurologists, as well as psychometric testing, I believe that surgeons should be cautious in using RCP as a means of extending DHCA. If it is possible to extrapolate from the pediatric experience with DHCA, periods of DHCA greater that 30 to 45 minutes in length at a tympanic membrane temperature of 15°C should be used with great caution.

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To the Editor:

We thank Dr Jonas for his informative response regarding our article and appreciate his contributions to this field in pediatric cardiac surgery.

We did have neurologists or radiologists involved in this study. They did preoperative or postoperative neurologic examinations of our patients or reviewed the radiologic images. We must apologize for not including their names among the authors of our paper and for not stating their con-

tribution in the methods section. We acknowledge that the inference that "a DHCA + RCP period of up to 80 minutes under a nasopharyngeal temperature of 18°C is safe" in our experience is rather anecdotal and cannot be totally substantiated. However, if we divide the patients into 2 groups—those who had DHCA + RCP for less than 60 minutes and those in whom this period lasted 60 minutes or more—we cannot find any difference in the incidence of mortality, stroke, and transient neurologic dysfunction. However, it is obvious that we should reduce the period of DHCA as much as possible, even in patients who require a complex repair of the aortic arch.

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Reconstruction of the aortic valve with autologous pericardium: An experimental study

To the Editor:

Valve repair has several advantages over prosthetic valve replacement, including low morbidity and mortality and lower risk of thromboembolism, hemorrhage, and septic endocarditis. Different techniques of mitral valve repair have been described, with good results.[1] Unfortunately, the results of aortic valve repair are not as favorable. In this report I describe a new technique of aortic valve reconstruction with an autologous pericardial patch, which my colleagues and I have used.

In 5 cadaver hearts, autologous pericardium was fixed in 0.6% glutaraldehyde solution for 10 minutes.[2] An aortotomy was used. Pericardium was cut to simulate the dimensions of the valve to be repaired on the basis of the concept of the aortic root geometry as a truncated cone.[3] The diameter at the highest point of attachment of the leaflets (sinotubular diameter) is about 20% less than the diameter at the inlet (surgical anulus diameter). During systole the sinotubular diameter increases while the inlet diameter decreases, changing the root geometry from conical to cylindrical. The reverse occurs in diastole, at which time the leaflets tilt toward the ventricle.[4]

The noncoronary cusp is fashioned first. It is measured at its attachment line and height with a soft wire (Fig 1, *A*) and the measurements are marked on the pericardium (Fig 1, *B*). The resulting "a-b" is the length at the sinotubular circumference but not the true length of the cusp. Therefore, we lengthened it to the size of its projection into the surgical (inlet) circumfer-

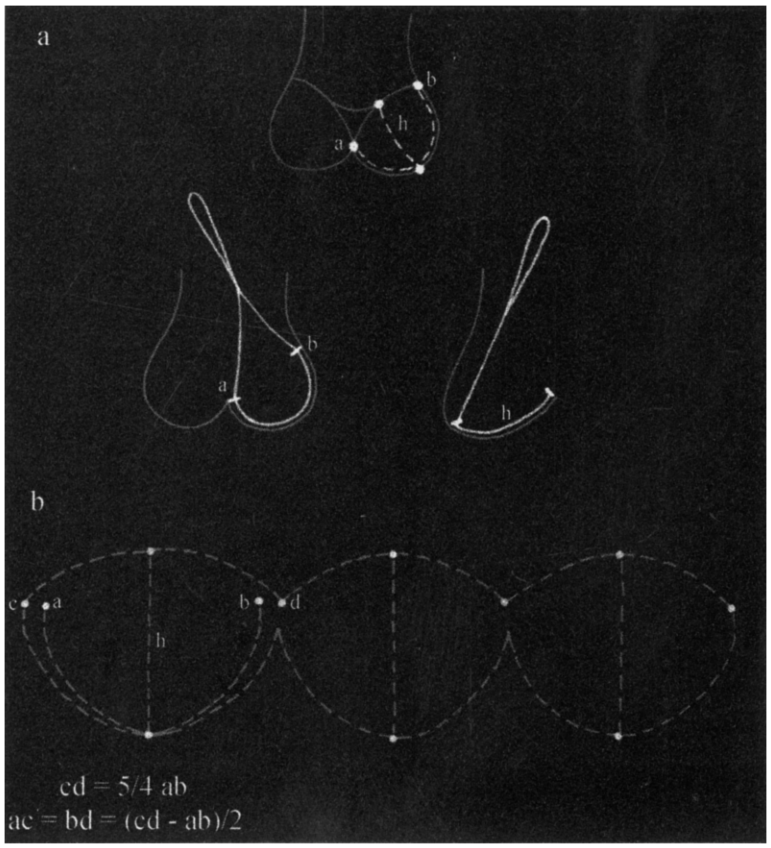

Fig 1. Cusp size measurements and fashioning of the pericardial patch. **A,** Taking of the noncoronary cusp attachment line and height measurements with the soft wire. **B,** Marking of the sizes on the pericardium and fashioning of the pericardial patch.